The Effects of Spinal Anesthesia On the Fetal Heart Rate

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• **The effect of spinal anesthesia on fetal heart rate is due to maternal hypotension and subsequent fetal hypoxia. Maternal hypotension of 80 mm of mercury for five minutes almost always results in hypoxic fetal bradycardia. This bradycardia is gradual in onset, and may be preceded by a short period of fetal tachycardia. There is a lag in the return of fetal heart rate to normal after maternal blood pressure has normalized. Similar bradycardia has been observed in maternal syncope unassociated with anesthesia. Maternal hypotension should be prevented, and if it occurs should be corrected early. Administration of a vasopressor drug is the treatment of choice, with oxygen and fluids as indicated.**

THE PURPOSE of the present day study is to learn what effect spinal anesthesia has on the fetal heart rate as recorded by the fetal electrocardiogram, and what aspect of the anesthesia is responsible for the effect. It is to be noted that the fetal electrocardiogram is being used more and more widely as an indication of the intra-uterine condition of the fetus. Until about 1952 the fetal heart rate was the primary guide to an abnormal fetal physiologic condition. A slow, fast or irregular heart rate, especially during the period of labor, was the principal indication of the condition of the fetus. Management of labor was based largely upon these observations.

During the past decade thousands of case studies have broadened and refined the usefulness of the fetal electrocardiogram. One recent report[9] alone is based on 4,500 fetal electrocardiogram studies. The technique has been helpful in early diagnosis of pregnancy, presentation, multiple pregnancy, death-in-utero and conditions such as Rh incompatibilities, toxemia, ante-partum bleeding, heart disease and metabolic diseases such as diabetes and thyroid dysfunction.

TECHNIQUE

Customarily, monitoring of the fetal heart is done with a fetal stethoscope applied intermittently upon the abdominal wall. Obviously this technique misses events occurring during the non-monitored periods. Attempts to monitor continuously with microphones strapped to the abdominal wall have not been very successful, due to the large amount of noise introduced from patient movement and other causes. Therefore, attempts were made to electrically monitor the fetal heart rate by use of electrocardiogram electrodes applied to the abdomen. Four sources of "noise" which proved troublesome in recording were: apparatus noise, environmental noise, skin-electrode junction noise, and patient motion noise.

Figure 1 shows three types of electrodes in diagrammatic fashion. It is to be noted that potentials from the abdomen are in the order of 5 to 25 microvolts. However, noise is in the order of 5 microvolts. Hence important data is submerged in worthless random noise. As was previously mentioned, a portion of the offending noise results from motion at the skin-electrode junction. The vaginal electrode shown in Figure 2 is better for its leads are not enclosed in plastic and it is freer to move with the patient. Vaginal electrodes permit pickup of fetal QRS amplitudes of 50 to 150 microvolts —a tremendous gain in signal strength.

As might be expected, signals from all these electrodes contain impulses from the maternal as well as from the fetal heart. In this application maternal electrocardiograms are unwanted and constitute electrical noise. As might be further expected with the use of vaginal electrodes applied to the presenting part, the signal from the maternal heart is of relatively low amplitude as compared with that from the fetal heart. Attenuation of noise is possible with suitably designed filter and amplifier circuitry, but with some loss of signal. Figure 3 shows a two channel recording of a filtered and unfiltered signal. The baseline of the filtered tracing is much "cleaner." The tracing is suitable for feeding into a cardiotachometer and permits an accurate "beat-to-beat" neon display of the fetal heart rate. Multichannel recording permits this value to be recorded

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along with maternal blood pressure and uterine pressure studies.

Techniques are currently being developed in our hospital for averaging correlated signals occurring in random noise. The result is a linear summation of the correlated signal and a quadratic summation of the uncorrelated noise. The end result is a dramatic improvement in signal-to-noise ratio. The present study, however, employs the fetal electrocardiogram as a method of obtaining an accurate "beat-to-beat" evaluation of the fetal heart rate.

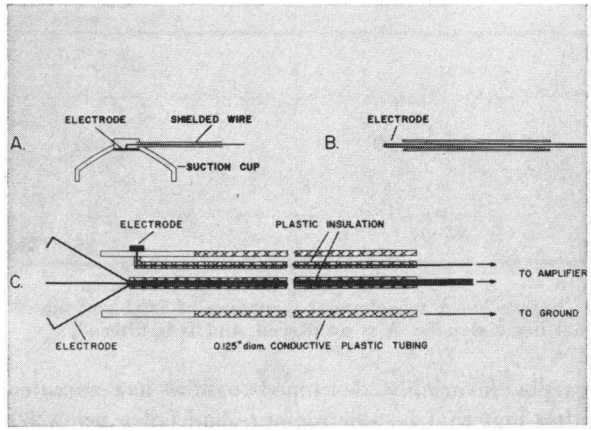

Figure 1.—Diagrams of three types of electrocardiogram electrodes used for constant monitoring of fetal heart rate. A, section cup type; B, needle type; C, vaginal type with its leads enclosed in a ⅛ inch diameter plastic tube.

DISCUSSION

Ebner, Barcohena and Bartoshuk[2] concluded from their study that maternal hypotension is responsible for fetal bradycardia in spinal anesthesia. They found that the incidence of bradycardia was proportional to the severity and duration of maternal hypotension. A systolic pressure of 60 mm of mercury for four minutes resulted in fetal bradycardia. If normal tensions were restored within that time, fetal bradycardia did not occur. When maternal systolic pressure did not drop below 70 mm of mercury, fetal bradycardia was unlikely.

Hingson and Hellman[4] said that fetal brady-

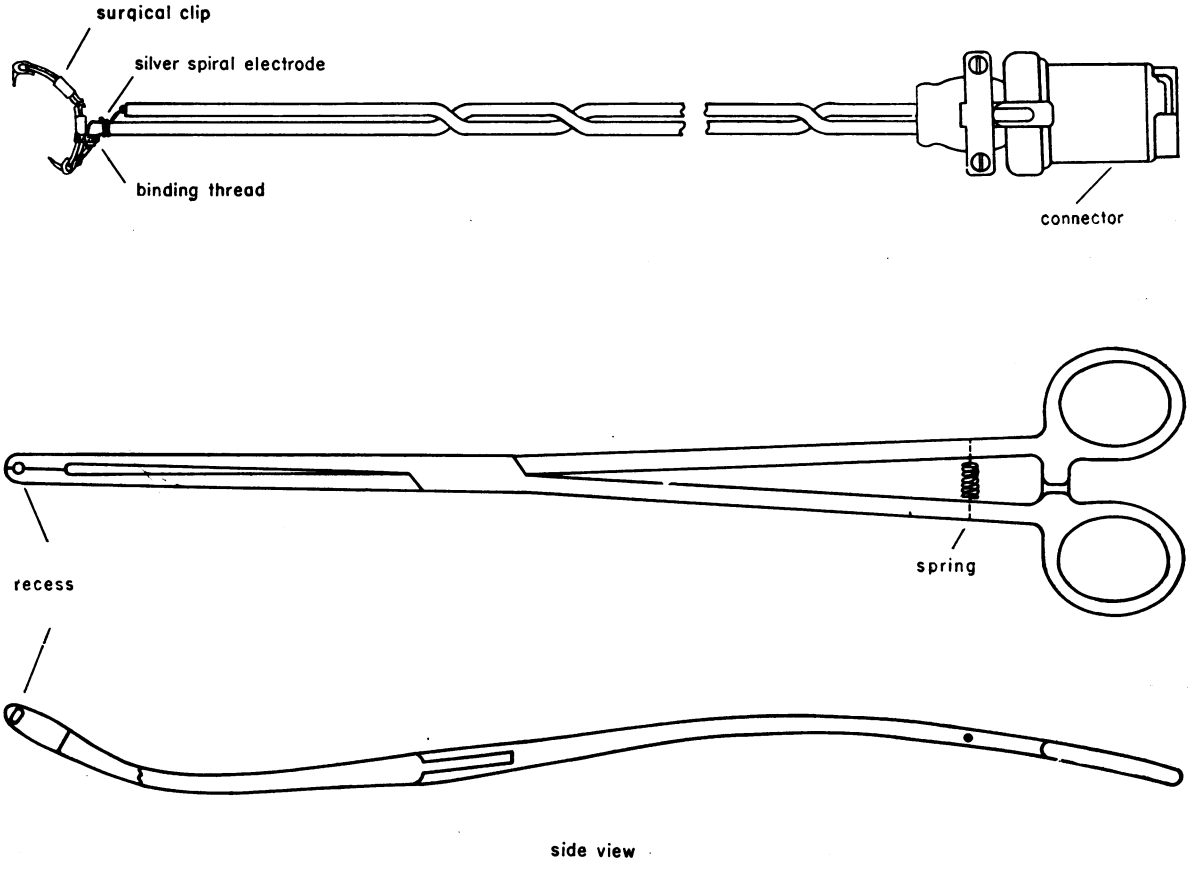

Figure 2.—Vaginal electrode and instrument for placing on presenting part.

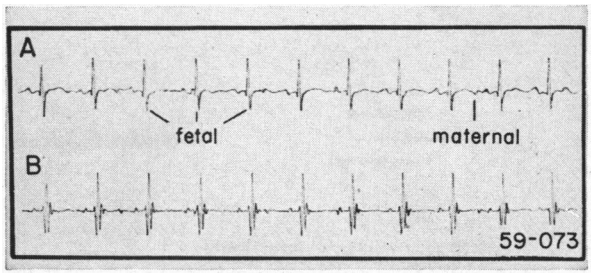

Figure 3.—A two-channel recording of fetal and maternal heart signals: A is unfiltered, and B is filtered.

cardia invariably developed within five minutes after maternal systolic pressure had fallen below 80 mm of mercury.

Wylie[10] expressed belief that maternal hypotension may lead to rapid fetal death. He wrote that maternal hypotension of 80 mm or below for five minutes would lead to fetal hypoxia.

Dance and Ward[1] stated that hypotension in the mother is a constant threat and may cause hypoxia in the fetus.

After an extensive study of fetal bradycardia in 1959, Hon[5,6] made several conclusions, among which are the following four:

1. Adequate intervillous space blood flow and oxygenation are probably related to a normal differential between intramyometrial and maternal blood pressure, as well as sufficient relaxation between uterine contractions. This fact is of definite clinical importance in a patient receiving an oxytocic agent[3] for the induction of labor. Excessively rapid administration of the agent, leading to three 60 to 80 second contractions in 6 to 7 minutes resulted in fetal tachycardia preceding and following an episode of fetal bradycardia. During this period of uterine hypertonicity the intervillous space may be physiologically isolated and lead to a depletion of fetal oxygen reserve. When maternal hypotension is an additional complicating factor, as may occur with conduction anesthesia, serious fetal hypoxia can result.

2. The electrocardiographic pattern of fetal bradycardia noted with strong uterine contractions is similar to that seen with maternal hypotension. There is a U-shaped pattern, as may be seen in Figure 4. Increasing fetal hypoxia is indicated by a progressively longer, and more irregular U pattern.

3. Fetal bradycardia associated with umbilical cord compression is probably due to vagotonia. Hon and co-workers[7] also reported observing, in 31 mothers to whom they gave atropine, a decided change in the fetal heart rate pattern of bradycardia due to cord compression at delivery. (See Figure 5.)

4. The first indication of mild fetal hypoxia may be a sustained fetal tachycardia. This is not cause for immediate delivery. Three cases of sustained fetal tachycardia during maternal hypothermia were observed without apparent injury to the fetus. Hypoxic bradycardia is due to the hypoxic fetal myocardial depression, and begins near the *end* of a contraction. A similar pattern has also been observed in maternal syncope unrelated to anesthesia.

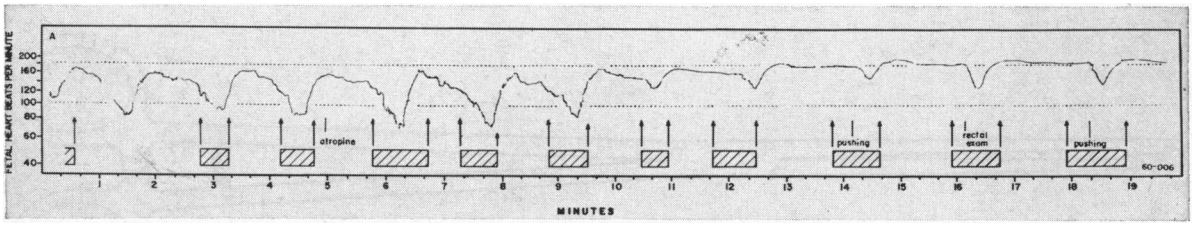

Figure 4.—Ordinate showing fetal heart rate in beats per minute and shaded areas showing period of contraction of uterus. Note the U-shaped curve which began and ended within the duration of the contraction. Contrast with Figure 5.

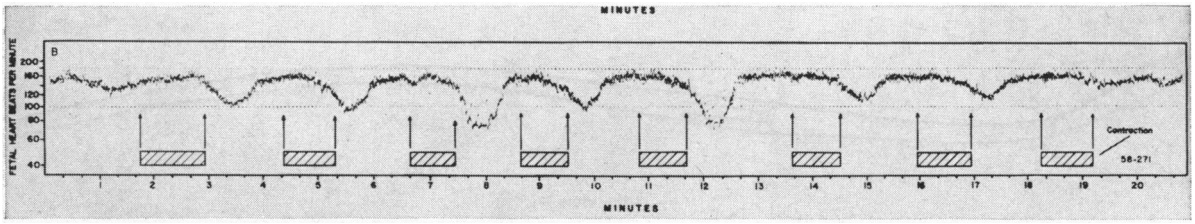

Figure 5.—The U-shaped curve of vagotonia developing late in contraction and extending well into the resting period of uterine relaxation. Contrast with Figure 4.

PRESENT STUDY

A search was made of 65 anesthetic records of mothers having fetal electrocardiographic studies during delivery under spinal anesthesia. It was not felt necessary to report on varying doses of *Pontocaine®*, given by residents, in this preliminary study. Although twelve of the 65 records showed a blood pressure drop to 80 mm of mercury or below, no fetal heart rate abnormalities occurred in any of these cases, indicating prompt and efficient correction of maternal hypotension.

TREATMENT

The treatment of fetal bradycardia following spinal anesthesia is essentially the treatment of maternal hypotension. Accurate and frequent determination of blood pressure is essential. Apparatus for automatic, periodic checking of maternal blood pressure is now available. The use of a vasopressor drug is probably of first importance in treatment. One hundred per cent oxygen is of value, but has less effect in returning the maternal blood pressure to normal than does a pressor agent. Ephedrine sulfate is at present thought to be the drug of choice. Methoxamine hydrochloride is thought to cause constriction of placental tissue and to aggravate fetal hypoxia. Selection of vasopressor agents should be made with this in mind. Fluid therapy should be instituted as indicated.

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